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=> S (Rab11A) (8A) (HIV-1)
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L3 ANSWER 1 OF 3 MEDLINE on STN DUPLICATE 1
AN 2008337671 MEDLINE
DN PubMed ID: 18406652
TI Statin-induced inhibition of HIV-1 release from latently infected U1 cells
reveals a critical role for protein prenylation in HIV-1 replication.
AU Amet Tohti; Nonaka Mizuho; Dewan Md Zahidunnabi; Saitoh Yasunori; Qi
Xiaohua; Ichinose Shizuko; Yamamoto Naoki; Yamaoka Shoji
CS Department of Molecular Virology, Graduate School of Medicine, Tokyo
Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8510,
Japan.
SO Microbes and infection / Institut Pasteur, (2008 Apr) Vol. 10, No. 5, pp.
471-80. Electronic Publication: 2008-01-20.
Journal code: 100883508. ISSN: 1286-4579.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200807

ED Entered STN: 28 May 2008
 Last Updated on STN: 16 Jul 2008
 Entered Medline: 15 Jul 2008

AB Latent infection of human immunodeficiency virus type 1 (HIV-1) represents a major hurdle in the treatment of acquired immunodeficiency syndrome (AIDS) patients. Statins were recently reported to suppress acute HIV-1 infection and reduce infectious virion production, but the precise mechanism of inhibition has remained elusive. Here we demonstrate that lypophilic statins suppress HIV-1 virion release from tumor necrosis factor alpha-stimulated latently infected U1 cells through inhibition of protein geranylgeranylation, but not by cholesterol depletion. Indeed, this suppression was reversed by the addition of geranylgeranylpyrophosphate, and a geranylgeranyltransferase-1 inhibitor reduced HIV-1 production. Notably, silencing of the endogenous Rab11a GTPase expression in U1 cells by RNA interference destabilized Gag and reduced virion production both in vitro and in NOD/SCID/gammac null mice. Our findings thus suggest that small GTPase proteins play an important role in HIV-1 replication, and therefore could be attractive molecular targets for anti-HIV-1 therapy.

L3 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 2
 AN 2006111648 MEDLINE
 DN PubMed ID: 16497224
 TI The pericentriolar recycling endosome plays a key role in Vpu-mediated enhancement of HIV-1 particle release.
 AU Varthakavi Vasundhara; Smith Rita M; Martin Kenneth L; Derdowski Aaron; Lapierre Lynne A; Goldenring James R; Spearman Paul
 CS Department of Pediatrics, Vanderbilt University School of Medicine, Nashville, TN 37232-2581, USA.
 NC DK38063 (United States NIDDK NIH HHS)
 DK48370 (United States NIDDK NIH HHS)
 P30 AI1054999 (United States NIAID NIH HHS)
 R01 AI058828 (United States NIAID NIH HHS)
 SO Traffic (Copenhagen, Denmark), (2006 Mar) Vol. 7, No. 3, pp. 298-307.
 Journal code: 100939340. ISSN: 1398-9219.
 CY Denmark
 DT (COMPARATIVE STUDY)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 LA English
 FS Priority Journals
 EM 200605
 ED Entered STN: 28 Feb 2006
 Last Updated on STN: 25 May 2006
 Entered Medline: 24 May 2006

AB The HIV-1 accessory gene product Vpu is required for efficient viral particle release from infected human cells. The mechanism by which Vpu enhances particle assembly or release is not yet defined. Here, we identify an intracellular site that is critical for Vpu-mediated enhancement of particle release. Vpu was found to co-localize with markers for the pericentriolar recycling endosome. Expression of dominant negative mutants of Rab11a and myosin Vb that disrupt protein sorting through the recycling endosome abrogated the ability of Vpu to augment particle release. Remarkably, the effects of blocking recycling endosome function on HIV particle release were demonstrable only in human cell lines known to be responsive to Vpu, while no effect on particle release was seen in African green monkey cells. Inhibition of recycling endosome function in human cells also blocked the ability of HIV-2 envelope to enhance particle release. These studies indicate that Vpu and HIV-2 envelope glycoprotein enhance particle release via a common

mechanism that requires the activity of the pericentriolar recycling endosome.

L3 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
AN 2005:1075825 CAPLUS
DN 143:360061
TI G proteins RAB9A and RAB11A playing a role in viral infection processes as targets for prevention of infectious disease
IN Hodge, Thomas W.; McDonald, Natalie J.; Rubin, Donald; Shaw, Michael W.; Sanchez, Anthony; Murray, James L.
PA The Government of the United States of America as Represented by the Secretary, Department of Health and Human Services Centers for Disease Control and Prevention, USA; Vanderbilt University
SO PCT Int. Appl., 105 pp.
CODEN: PIXXD2

DT Patent
LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	WO 2005092924	A2	20051006	WO 2005-US6396	20050224
	WO 2005092924	A3	20060511		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
	RW:	BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
	AU 2005226779	A1	20051006	AU 2005-226779	20050224
	CA 2557426	A1	20051006	CA 2005-2557426	20050224
	EP 1723177	A2	20061122	EP 2005-758743	20050224
	R:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU			
	JP 2008506356	T	20080306	JP 2007-500800	20050224
	EP 1958964	A2	20080820	EP 2008-5922	20050224
	EP 1958964	A3	20090107		
	R:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR			
	US 20070087008	A1	20070419	US 2006-590844	20060824
	IN 2006KN02795	A	20070601	IN 2006-KN2795	20060925
PRAI	US 2004-547328P	P	20040224		
	EP 2005-758743	A3	20050224		
	WO 2005-US6396	W	20050224		

AB The G proteins RAB9A and RAB11A that play important roles in the regulation of cellular processes are found to play a role in processes used by pathogens in the infection of host cells and therefore may be targets for the prevention and treatment of infection. Exemplary pathogens include those that use a lipid raft. SiRNAs against a series of genes were tested for the effects on the infection of HIV-1 in JC53-BL cells. Lowered efficiency of infection were associated with lowered levels of mRNA for RAB9A and RAB11A. SiRNAs against proteins modulating RAB9A activity also limited the ability of HIV-1 to infect cells.

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